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The Case | Renal failure after percutaneous closure of a perivalvular leak

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Figure 1 | Red urine.

A 71-year-old man with a history of congestive heart failure from valvular heart disease who had previously received aortic and mitral valve replacements was admitted with progressively worsening mitral perivalvular leak. He was judged to be a poor operative candidate and underwent percutaneous closure of the leak utilizing Amplatzer occluder device. Post-procedure, he was noted to have red urine (Figure 1) and his creatinine increased from 1.3 to 1.7 mg/100 ml. Enalapril and furosemide were stopped but the creatinine kept rising until it reached 2.8 mg/100 ml on the sixth day after valve closure. Hemoglobin had also dropped from 13.3 g/100 ml on admission to 10 g/100 ml. Urine analysis showed 2+ protein, 3+ heme, 23 WBC/HPF, 7 RBC/HPF, and 6–15 granular casts/HPF. A renal ultrasonogram was normal.

What is the cause of his renal failure?
How will you confirm the diagnosis?

SEE NEXT PAGE FOR ANSWERS

The Diagnosis | **Hemoglobinuria**

Lactate dehydrogenase level was 3347 U/l. Liver function tests showed an elevated aspartate aminotransferase level of 157 U/l and an indirect bilirubin of 3.6 mg/100 ml. Schistocytes were noted on the peripheral smear. Patient was started on intravenous dextrose 5% with 150 meq. bicarbonate/l drip at 75 ml/h. He also received darboepoietin, iron sulfate, folic acid, and *N*-acetyl cysteine. Hemogloblin dropped to 7.7 g/100 ml and he required red blood cells transfusions. Creatinine increased further to 3.9 mg/100 ml, and there was persistent hemoglobinuria. A transesophageal echo was performed showing moderate, possibly severe mitral regurgitation with the flow going between the amplatzer device and the sewing ring of the bioprosthetic valve, resulting in abnormal rocking of the prosthesis. Patient declined open mitral valve replacement and was transferred back to his referring hospital.

We describe a case of renal failure caused by intravascular hemolysis after percutaneous closure of mitral perivalvular leak with Amplatzer device. Intravascular hemolysis secondary to mechanical and bioprosthetic valves and patent ductus arteriosus closure devices have been well described in the literature. Distinct patterns of regurgitant flow associated with high shear stress were thought to cause hemolysis with intracardiac devices, whereas the presence of residual ductal flow was thought to cause hemolysis after percutaneous closure of patent ductus arteriosus especially with Amplatzer occluders. Recently, hemolytic anemia has been reported subsequent to percutaneous closure of traumatic ventricular septal defect using Amplatzer device, which required multiple blood transfusions and finally surgical removal of the device.[1] A review of 14 procedures performed between 2001 and 2004 on percutaneous closure of prosthetic paravalvular leaks involving both mitral and aortic valves showed that two patients had residual leaks and persistent anemia requiring transfusions at 1-year follow-up.[2] A more recent study involving 11 patients with perivalvular leaks who underwent percutaneous closure using the Amplatzer occluder resulted in worsening hemolysis in four patients, improvement in four, and no change in two patients, concluding that the Amplatzer occluder should be reserved for poor surgical candidates.[3] None of the patients in these two studies developed renal failure.

Even though kidney dysfunction has been rarely described following percutaneous closure devices, we believe that hemolysis should be considered as part of the differential in any patient who presents with renal failure after prosthetic device placement.

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